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Issue Date: 14 January 2003

BRB No. 99-0710 BLA
Case No. 1996-BLA-1622

In the Matter Of

BERNICE HYSLOP (Widow of
HUGH HYSLOP)
Claimant,
v.

OLD BEN COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS,
Party-in-Interest.

DECISION AND ORDER ON REMAND - AWARDING BENEFITS

Claimant appealed the Decision and Order - Denying Benefits as issued by Administrative Law Judge Donald W. Mosser on a miner's claim and a survivor's claim filed pursuant to the provisions of Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 *et seq. (*the Act). In its Decision and Order issued on September 27, 2000, the Benefits Review Board (the Board) affirmed in part and vacated in part, and remanded this case to the administrative law judge for further consideration consistent with the Board's decision.

The Board affirmed the finding that the medical evidence did not establish complicated pneumoconiosis pursuant to 20 C.F.R. Sections 718.202(a)(3) and 718.304(c). It also affirmed the finding of thirty-eight years of coal mine employment. The Board noted that the administrative law judge had found the autopsy prosector's report to be the most reliable evidence, and relying upon same, had determined that the Miner had established pneumoconiosis pursuant to 20 C.F.R. Section 718.202(a)(2), a finding it affirmed.

The Board vacated the finding, however, made pursuant to 20 C.F.R. Section 718.204(b). Specifically, Judge Mosser had found that the opinions of Drs. Tuteur, Crouch, Naeye and Wiot established that a separate, non-coal mine employment disease, idiopathic pulmonary fibrosis, caused the miner's total disability. The Board held that the administrative law judge did not provide

adequate reasons for crediting these medical opinions, noting that the administrative law judge relied upon Dr. Tuteur's opinion, as supported by those of Drs. Crouch, Naeye and Wiot. The Board explained as follows:

> The record indicates that Dr. Tuteur, who is Board-certified in Internal Medicine and Pulmonary Disease, reviewed the medical evidence and concluded that the miner was disabled by and died due to idiopathic pulmonary fibrosis superimposed upon preexisting chronic obstructive pulmonary disease due to smoking. Employer's Exhibits 2, 23. In contrast, Dr. Cohen, who is also Board-certified in Internal Medicine and Pulmonary Disease, reviewed the medical evidence and opined that the miner's anthracosis, pulmonary fibrosis, and emphysema were all related to coal dust exposure and contributed to the miner's total disability and death. Claimant's Exhibit 1. In crediting Dr. Tuteur's opinion, the administrative law judge stated that Dr. Tuteur's credentials were "more impressive."

The Board agreed with the Claimant's argument, that an explanation for finding Dr. Tuteur's credentials more impressive was not provided. The Board also questioned the administrative law judge's finding that the report of Dr. Tuteur was better reasoned, since the only reason identified by the administrative law judge for that conclusion was that Dr. Cohen dismissed the miner's heart disease as a potential cause of disability or death, which reason was not supported by substantial evidence. Accordingly, the findings pursuant to Section 718.204(b) were vacated and the matter remanded for a reweighing of the relevant evidence to determine whether pneumoconiosis was a contributing cause of the miner's total disability. Given that the same reasons were cited by the administrative law judge to find that pneumoconiosis did not hasten death, the findings pursuant to that section were also vacated, with instructions that the medical evidence be re-weighed in order to determine whether pneumoconiosis hastened the miner's death.

Employer filed a timely motion for reconsideration, which was denied by the Board on April 24, 2001. This matter was then remanded to this Office and by Order dated August 15, 2001, all parties were notified that this matter was being reassigned due to the fact that Judge Mosser was no longer with the Office. The parties were given thirty days in which to submit briefs on remand. Employer and Claimant both submitted briefs.

Issues

1. Whether pneumoconiosis was a contributing cause of the Miner's total disability.

2. Whether the Miner's death was hastened by pneumoconiosis.

3. Whether the Miner was and/or the Claimant is entitled to benefits under the Act.

Applicable Law and Regulations

Because these claims were made after March 31, 1980, the effective date of Part 718, they must be adjudicated under those regulations.

The Department of Labor has amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001 and are found at 20 C.F.R. Parts 718, 722, 725 and 726. All references are to the new regulations unless noted otherwise. In its decision, the Board cited to the old regulations.

The case law of the United States Court of Appeals for the Seventh Circuit, within whose jurisdiction this case arises, is applicable in this case.

DISCUSSION

Medical Opinion Evidence

In its decision, the Board discussed the reports of Drs. Tuteur, Crouch, Naeye, Wiot and Cohen. There are other medical reports of record, as detailed below.

Dr. Krishna Murthy examined the Miner on March 25, 1988. (DX 67). A cigarette smoking history of forty to fifty pack years was noted, the Miner having quit smoking fourteen years ago. Forty-two years of coal mine employment was also recorded. Based upon his examination, which included the taking of a chest x-ray and pulmonary function testing, Dr. Murthy diagnosed interstitial lung disease of reticulo-nodular variety. Dr. Murthy explained as follows:

> This is probably idiopathic in origin, however in view of the history of exposure to coal dust I would suspect this may represent coal worker's pneumoconiosis. Historically, this may be due to a combination of previous cigarette smoking and coal worker's pneumoconiosis. Since there has been a progression in symptoms I would like to rule out any infectious process or malignant process in this patient. Certainly, one has to consider and exclude sarcoidosis in stage III and possibly collagen vascular disease.

On June 20, 1988, Dr. Daniel J. Combs examined the Miner. (DX 60, 61). Dr. Combs performed an examination which included the taking of histories, a chest x-ray, pulmonary function study and blood gas testing. Dr. Combs found the Miner to be suffering from bronchiectasis secondary to smoker's bronchitis. Dr. Combs found the etiology of the diagnosed condition to be previous pulmonary

infection and environmental pollutants including cigarette smoke. A smoking history of one to one and a half packs of cigarettes per day from the age of 18 years until 1973 was recorded. Dr. Combs found the Miner unable to return to his coal mine work because of the bronchiectasis. "And pneumoconiosis" is added to that last sentence, however, it appears to be in a different handwriting than the rest of the report, and I do not find it to be reliable. Pneumoconiosis is not listed in the typed report submitted by Dr. Combs on August 1, 1998. (DX 61). Dr. Combs noted that bronchiectasis accounted for 60% of the Miner's disability and smoker's bronchitis accounted for 30%.

By letter dated September 21, 1988, Dr. Paul Wheeler stated that he had received a chest x-ray dated June 16, 1988. (DX 68). Dr. Wheeler stated that the Miner's lung disease had developed and progressed over the last four years. In his opinion, the natural history of this disease strongly favored sarcoid or tumor as opposed to pneumoconiosis. In his opinion, it was extremely unlikely that silicosis or coal worker's pneumoconiosis caused the present lung disease.

Dr. Jeff W. Selby examined the Miner on September 23, 1988. (DX 68). Dr. Selby recorded coal mine employment from 1943 until retirement at the age of sixty-five years. Dr. Selby also recorded a cigarette smoking history of one to one and a half packs per day for approximately 45 years. Based upon his examination, which included the taking of a chest x-ray, pulmonary function test and blood gas study, as well as a review of medical records, Dr. Selby opined that the Miner had a severe pulmonary abnormality. He found that it did not appear to be due to pneumoconiosis, but rather, due to a severe inflammatory process that most likely was idiopathic pulmonary fibrosis. Dr. Selby stated as follows:

> Having the opportunity to review the outside reports and workup separated by four years with the obvious x-ray abnormalities now present as compared to my own interpretation four years ago, it is apparent and exceedingly obvious that this is not coal worker's pneumoconiosis and indeed, is something much more serious and severe.

In a letter dated March 19, 1991, Dr. Combs stated that the Miner had had an x-ray dated February 12, 1991, revealing pneumoconiosis category 2/3 Q/T. (DX 74). Dr. Combs also reviewed the blood gas study and lung exam results. In his opinion, the Miner's clinical condition was compatible with pneumoconiosis secondary to exposure to coal dust.

The Miner died on September 3, 1994. (DX 2A). Dr. Terry Gelhausen listed the cause of death as acute myocardial infarction and pneumoconiosis - chronic acute pneumonia.

An autopsy was performed on September 4, 1994, by Dr. John A. Heidingsfelder. (DX 3A, 4A). Based upon same, Dr. Heidingsfelder

provided an autopsy report which listed the following Gross Anatomic Diagnosis: (1) increased pulmonary weight; (2) complete right pleural fibrosis adhesion; (3) pleural fibrous plaque, diffuse pattern, smooth, parietal pleura, left side, right-sided diffuse irregular pattern of fibrosis and residual fibrous adhesions; (4) pulmonary anthracosis, marked diffuse pattern;(5) marked anthracosis, peritracheal and peribronchial lymph nodes; (6) extensive white to cream colored mucous accumulation, trachea, large and small bronchi, bilaterally; (7) moderate trenching of tracheal and mainstem bronchi, mucosal surfaces, bilaterally; (8) pulmonary emphysema, diffuse parenchymal pattern, marked, bilaterally; (9) subpleural and parenchymal fibrosis; interstitial fibrosis and anthracosis, moderate, bilaterally; (10) multifocal regions of nodular consolidation with purulent mucus accumulation consistent with bronchopneumonia (lower lobes more severe than upper lobes); (11) cardiomegaly with moderate left ventricular hypertrophy; (12) right ventricular hypertrophy; (13) pipestem calcification of left anterior descending coronary artery with focal 50-60% narrowing by calcific plaque; moderate aortic atherosclerotic calcific placques. In his opinion, the findings were consistent with environmental lung disease (Black Lung disease). Multiple biopsies were taken on bilateral lung tissue, heart, trachea and lymph node tissues.

By report dated December 29, 1994, Dr. Gelhausen stated that he had been the miner's attending physician since February 6, 1993. (DX 4A). Dr. Gehlhausen pointed out that the Miner's past history included work at the tipple, where the amount of coal dust is known to be quite high, as well as a smoking history of one to one and a half packs of cigarettes per day from the age of eighteen years until 1973. Dr. Gelhausen also reviewed the autopsy findings. He concluded as follows:

> Prior to this patient's death his health was extremely compromised and his entire livelihood was directed by his respiratory failure. He was almost entirely wheelchair bound for the last year of his life and was certainly unable to pursue any outside interests. Certainly this patient had been totally disabled from pneumoconiosis prior to his death. The patient had been battling recurrent lung infections and, indeed, had a pneumonia at the time of his death. The excessive pressure on his heart due to the pneumoconiosis and pneumonia undoubtedly led to his acute and sudden death due to myocardial infarction.

In a follow-up report dated January 10, 1995, Dr. Murthy stated that the Miner was first seen by him on March 25, 1988. (DX 9A). At the time, Dr. Murthy saw that the Miner could not climb a flight of stairs without getting short of breath. A cigarette smoking history lasting from the age of eighteen years to sixty-three years was recorded, the Miner having smoked one pack per day for this forty-five year period. A total of thirty-nine years of coal mine employment was also recorded. Dr. Murthy pointed out

that his last contact with the Miner was in March of 1994, at which time the Miner continued to be short of breath, even with mild exertion. The Miner was using supplemental oxygen at two liters per minute continuously. At that time, Dr. Murthy's impression included diffuse interstitial lung disease of fine reticular as well as reticular-nodular variety, hypoxemia secondary to interstitial lung disease, cor pulmonale and coal worker's pneumoconiosis and chronic bronchitis. Dr. Murthy stated that after the Miner's death in early September, an autopsy was performed. At autopsy, the pulmonary parenchyma, in particular the right lung, revealed diffuse black discoloration and there were areas of interstitial fibrosis. Dr. Murthy reviewed additional findings upon autopsy and concluded as follows:

> In essence, Mr. Hyslop had coal worker's pneumoconiosis as is evidenced by the autopsy findings of multiple foci of nodular consolidation and black discoloration of the upper lung fields and peritracheal as well as peribronchial lymph nodes. He also had evidence of chronic bronchitis related to chronic cigarette smoking. It would appear, from the autopsy findings, that he had predominantly coal worker's pneumoconiosis which was most certainly a contributing cause of his death.

Dr. Murthy is board certified in internal medicine, pulmonology and critical care medicine.

Dr. John C. MacLennan submitted a report dated January 11, 1995. (DX 5A). Therein, he stated that he attended the Miner from August 2, 1994, to August 9, 1994. Based upon his review of the Miner's work and smoking histories, as well as his examinations of the Miner and his review of the autopsy findings, Dr. MacLennan opined that the Miner was permanently and totally disabled by Black Lung Disease in the final years of his life, and while it was not a direct cause of his death, it was a very significant contributing factor to his death. Dr. MacLennan explained that this was because "cor pulmonale and respiratory failure, both of which were brought on by Black Lung disease, put an additional strain on his heart." Dr. MacLennan is board-certified in internal medicine with a subspecialty in cardiology.

Dr. John A. Heidingsfelder reviewed ten microscopic H&E slides by report dated August 17, 1995. (DX 12A). Dr. Heidingsfelder is board-certified in anatomic and clinical pathology. He is also board-certified in forensic pathology. Based upon his review, Dr. Heidingsfelder found (1) pleural, subpleural and interstitial pulmonary fibrosis, marked; (2) pulmonary anthracosis, marked, associated with focal anthracotic macular lesions; (3) pulmonary emphysema, moderate to marked; (4) acute and chronic bronchitis; (5) pneumonia; (6) pulmonary fibrohyaline nodule formations; (7) parietal plural and epicardial surface of heart fibrous plaque formations; (8) lymph node showing marked anthracosis; and (9) heart showing patchy myocardial fibrous replacement and evidence of myocardial cell hypertrophy. In his opinion, the macroscopic and

microscopic findings revealed severe chronic lung disease consistent with environmental lung disease (Black Lung Disease).

On May 23, 1995, and in response to a request from the Department of Labor for a "Reasoned Medical Opinion," Dr. Sarah B. Long submitted a written statement, indicating that in order to document coal worker's pneumoconiosis, she needed the pathologist's report of microscopic examination.(DX 8A).

On February 13, 1996, and after reviewing the microscopic portion of the autopsy report, Dr. Long concluded that death was due to pneumoconiosis. (DX 13A). She stated as follows:

> The microscopic examination of the lung tissue is consistent with coal worker's pneumoconiosis. His death was due to respiratory failure and pneumonia. In my opinion the underlying coal worker's pneumoconiosis was a significant contributing cause in this man's death.

On April 11, 1996, Dr. Edmond C. Crouch submitted his pathology consultation report. (EX 1). Therein, Dr. Crouch recorded that he had reviewed ten glass slides with the corresponding autopsy report regarding the Miner. Dr. Crouch found histologic evidence of dust exposure, but no histologically discernable dust-related lung disease. In particular, he found that pathologic changes of coal worker's pneumoconiosis were not identified, finding no coal dust macules, micronodules, or nodules or focal emphysema. No silicotic nodules were observed. In his opinion, the findings were consistent with idiopathic pulmonary fibrosis. Dr. Crouch concluded that occupational coal dust exposure could not have caused any functional impairment or respiratory disability and could not have contributed to or otherwise hastened death from end stage pulmonary fibrosis. Dr. Crouch is board-certified in anatomic pathology.

Dr. Richard L. Naeye submitted a report dated April 22, 1997, having reviewed medical reports, the death certificate, autopsy report and ten glass slides of tissue removed at autopsy. (EX 2). Based upon his review, Dr. Naeye found the major abnormality present to be massive interstitial fibrosis that in many areas had developed into a dense fibrosis that had replaced almost all normal lung tissue. He found a small amount of black pigment in the lungs. In his opinion, none of the characteristic findings of coal worker's pneumoconiosis were present in the Miner's lungs. There were no micronodules in his lungs, no fibrous tissue or focal emphysema independently associated with the black pigment that was present, and the larger birefringent crystals that were admixed with the black pigment were presumably silicates which were non-toxic. Dr. Naeye found death to have been the result of a very advanced interstitial pulmonary fibrosis, superimposed acute lobular pneumonia and a myocardial infarction that led to progressive cardiac failure. None of these disorders were the consequence of occupational exposure to coal mine dust. "Being absent, CWP could not have prevented this man from doing hard

physical work and it did not hasten his death." Dr. Naeye is board-certified in anatomic and clinical pathology.

Dr. Peter G. Tuteur submitted a report on November 21, 1997, having reviewed medical records dating from 1976 to 1997, including the autopsy report and pathology consultation reports of Drs. Crouch and Naeye. (EX 3). Based upon the totality of the available medical data, Dr. Tuteur concluded that the Miner died at the age of 84 years "with and because of coal mine dust unrelated health problems." Those problems included recurrent acute bronchopneumonia superimposed on end-stage interstitial pulmonary fibrosis (UIP) with honeycombing features and arteriosclerotic heart disease associated with congestive heart failure, all superimposed on cigarette smoke-induced chronic obstructive pulmonary disease.

In his opinion, none of these conditions were in any way related to, aggravated by or caused by the inhalation of coal mine dust or the development of coal worker's pneumoconiosis. Dr. Tuteur stated that the autopsy evaluation confirmed the absence of fulfillment of criteria for coal worker's pneumoconiosis, and confirmed the presence of the "specific" diagnosis of usual interstitial pneumonitis so advanced as to form honeycomb lung. It was this condition, unrelated to the inhalation of coal mine dust and complicated by cigarette smoke-induced chronic obstructive pulmonary disease as well as arteriosclerotic heart disease leading to recurrent infectious bronchopneumonia that resulted in the Miner's ultimate death. Dr. Tuteur found no evidence that the Miner suffered from coal worker's pneumoconiosis during his life, or any other coal mine dust-induced process. Though Dr. Tuteur found that the Miner was clearly disabled during life, neither coal worker's pneumoconiosis, nor any coal mine dust-related disease process contributed to this disability in whole or in part. Dr. Tuteur is board-certified in internal medicine and pulmonary disease.

The deposition testimony of Dr. Naeye was taken on December 17, 1997. (EX 18). Dr. Naeye testified that in his opinion, when an individual has evidence of pneumoconiosis on autopsy but the chest radiographs tended to be interpreted as negative by B-readers, the prosector only removed the blackest things that he or she could find and, in essence, made a diagnosis of CWP which is more severe than was actually present. In his opinion, the finding of anthracotic pigment is not the same as a finding of coal worker's pneumoconiosis. If there is no fibrosis or focal emphysema associated with the black pigment found in the lungs then the black pigment itself will not cause any abnormality in lung function. Dr. Naeye reiterated his findings upon review of the evidence herein. He found massive interstitial fibrosis to be present. There was some black pigment in the Miner's lungs, however, it was only small in amount. There was fibrous tissue in some of the places where there was black pigment, however the fibrous tissue always extended far beyond the black pigment. This was why Dr. Naeye was able to conclude that the relationship between the two was not cause and effect. In his opinion, the black pigment had just "been there earlier and then this other

disease process came along, probably a viral infection in his lungs, and fibrous tissue was laid down."

Dr. Naeye stated that the black pigment which was found was in the usual places where one would expect to find it in a coal worker. There, was however, only a small amount. Classic macules and micronodules were not present in the Miner's lungs. As Dr. Naeye explained:

> The only places where there was some black pigment were areas where there had been tremendous damage in the lungs so the black pigment couldn't be transported to the lymphatics and other normal structures in the lung.

It was Dr. Naeye's conclusion that the Miner did not suffer from coal worker's pneumoconiosis "because none of the characteristic findings of coal worker's pneumoconiosis were present." He found no anthracotic micronodules or focal emphysema. Death was due to a combination of interstitial fibrosis which made many parts of the lungs non-functional with a superimposed acute lobular pneumonia which was very advanced and very severe in some areas, and the old myocardial infarcts. None of these conditions were related to coal mine dust exposure.

Upon reviewing the report of Dr. Heidingsfelder, Dr. Naeye stated his agreement that anthracosis was present, although in his opinion coal worker's pneumoconiosis was not present. He also found fibrous tissue to be present, pulmonary emphysema to be present, acute and chronic bronchitis to be present and pneumonia to be present, as those were set forth in Dr. Heidingsfelder's report. In fact, Dr. Naeye agreed with Dr. Heidingsfelder's assessment. He also stated his agreement with the assessment rendered by Drs. Crouch and Tuteur. When asked how he knew that coal dust did not cause the Miner's idiopathic interstitial pulmonary fibrosis, Dr. Naeye responded that it was "[b]ecause that's not a disease process that is produced by coal worker's pneumoconiosis."

The deposition testimony of Dr. Selby was taken on December 19, 1997. (EX 19). Dr. Selby testified that he examined the Miner in 1984 and in 1988, and that he also had the opportunity to review medical records of the Miner. Dr. Selby stated that when he examined the Miner in 1988, the pulmonary function studies were not consistent with something one would see with coal worker's pneumoconiosis since the Miner had been out of the coal mine for about ten years when he was seen in 1984. Dr. Selby explained:

> If this was coal worker's pneumoconiosis, it should have been very static or stable four years later and should have corrected for his age in regards to his pulmonary function testing....So this is a dramatic decline that should not at all be consistent with coal worker's pneumoconiosis.

Dr. Selby's review of chest x-rays in 1988 resulted in the opinion that there was an interstitial infiltrate consistent with an intrinsic inflammatory process rather than silicosis or any inhaled dust. Dr. Selby stated that his "highest choices" included idiopathic fibrosis or pulmonary fibrosis, allergic alveolitis, sarcoidosis, lymphangitic carcinoma or a variety of other infiltrated lung diseases. When asked to explain what an idiopathic pulmonary fibrosis is, Dr. Selby called it a "diagnosis of exclusion usually," as one "doesn't really know why an individual should develop it." When asked if anthracosis was the same as coal worker's pneumoconiosis, Dr. Selby replied that it was not. Anthracosis was black pigment in the lung which can result from cigarette smoking, living in a crowded urban area, working in a coal mine or any other carbonaceous inhalation condition. Coal worker's pneumoconiosis is a specific pathologic pattern usually associated with focal emphysema and the particular characteristics of scarring that is fairly specific to just coal worker's pneumoconiosis. After reviewing the autopsy information in conjunction with the two exams he himself performed on the Miner during his lifetime, Dr. Selby opined that the Miner had no contribution of coal mine dust to cause COPD or emphysema. In his opinion, the Miner did not suffer from coal worker's pneumoconiosis or any disease of the lungs caused or aggravated by his exposure to coal mine dust for nearly forty years. Death was due to idiopathic pulmonary fibrosis, longstanding, end stage.

In a deposition taken on March 30, 1998, Dr. Jerome Wiot, who is a B-reader and a board-certified radiologist, testified regarding the difference between coal worker's pneumoconiosis and idiopathic pulmonary fibrosis. (EX 22) In his opinion, the chest x-rays he reviewed revealed idiopathic pulmonary fibrosis, and not coal worker's pneumoconiosis. The former condition was not related to coal mine employment.

Dr. Robert Cohen submitted a report dated April 13, 1998. (CX 1). Dr. Cohen is board-certified in internal medicine and pulmonary disease. Dr. Cohen recorded the Miner's work and smoking histories, and reviewed the medical records from 1976 through the Miner's death and the autopsy report and consultative opinions on autopsy report and slides. Based upon his review, Dr. Cohen opined that the Miner did suffer from coal worker's pneumoconiosis. He based his opinion on the Miner's thirty-eight years of coal mine employment, his symptoms of chronic respiratory distress, the findings upon physical examinations beginning in 1980 when wheezing was heard, revealing numerous findings consistent with chronic respiratory disease, x-ray evidence, pulmonary function studies and autopsy evidence. It was his opinion that the Miner began to experience symptoms of dyspnea while still in the coal mines, and that he developed the beginnings of restrictive lung disease in 1979. His symptoms progressively worsened with acceleration beginning around 1986 or 1987.

According to Dr. Cohen the autopsy made it clear that the miner suffered from long term and chronic anthracosis as well as

silicoanthracosis which was associated with chronic and eventually massive pulmonary fibrosis. The Miner also had chronic bronchitis and cor pulmonale arising from his chronic pulmonary condition. It was Dr. Cohen's opinion that exposure to coal dust in over thirty-five years of work at the tipple "certainly contributed substantially to [the Miner's] anthracosis, silicoanthracosis, and pulmonary fibrosis. Coal dust along with his 45-65 pack year tobacco smoke exposure contributed to his emphysema and chronic bronchitis." Dr. Cohen concluded that the Miner was unable to perform his last coal mine duties, suffering from a severe and disabling pulmonary impairment. Death was due to severe end stage lung disease from pneumoconiosis, repeated pneumonias, right sided heart failure and a possible component of congestive heart failure. He did not think that an acute heart attack caused the Miner's death, and even if it did, it would only have been the "final pathway cause by the enormous stress of his end stage pulmonary process." Dr. Cohen concluded as follows:

> ...Mr. Hyslop's nearly 40 years of exposure to coal dust was significantly contributory to the development of his severe coal worker's pneumoconiosis, anthrasilicosis, pulmonary fibrosis, severe restrictive lung disease with diffusion impairment, and severely altered gas exchange. This pulmonary impairment was disabling, and ultimately lead to his death.

Dr. Cohen further stated that the autopsy revealed evidence of clumps of anthracotic pigment, which, in his opinion, is the "classic coal macule." Dr. Cohen found no doubt in his mind that the Miner's nearly forty years of exposure to coal dust was significantly contributory to the development of fibrosis and scarring in his lung. He added, "In fact, it makes little sense to attribute this patient's extensive pulmonary fibrosis to "idiopathic" or unknown cause, when he has nearly 40 years exposure to a known cause of fibrosis, coal dust."

Dr. Tuteur submitted a supplemental report on June 2, 1998. (EX 23). At that time, he had reviewed the report of Dr. Cohen, the deposition testimony of Drs. Naeye, Wiot and Selby and seventeen additional chest radiographs. Dr. Tuteur stated that this data continued to support the conclusions reached and expressed in his prior report. With regard to the opinion expressed by Dr. Cohen, Dr. Tuteur stated as follows:

> Reflecting on the basis of Dr. Cohen's opinion, one recognizes that Mr. Hyslop was exposed to sufficient amounts of coal mine dust to produce coal workers' pneumoconiosis in a susceptible host. The concentration of coal mine dust envisioned by Dr. Cohen is likely an overestimate since he believed that the tipple was located in an "enclosed building" and that visible dust can be equated with respirable dust. These latter two statements are incorrect.

In Dr. Tuteur's opinion, while the Miner did suffer from symptoms of chronic respiratory disease, these symptoms are not regularly associated with coal worker's pneumoconiosis, but with a cigarette-smoked induced chronic obstructive pulmonary disease. Dr. Tuteur found that Dr. Cohen had mis-characterized the autopsy evidence, as the "[c]areful competent microscopic evaluation of lung tissue failed to fulfill even rudimentary criteria for the diagnosis of coal worker's pneumoconiosis." It was Dr. Tuteur's opinion that the Miner did have a disabling pulmonary/respiratory condition which was cigarette-induced and in later life complicated by idiopathic interstitial pulmonary fibrosis. None of the Miner's disability was related in whole or in part to coal mine dust exposure, nor did it hasten or contribute to his death.

Total Disability Due to Pneumoconiosis

Judge Mosser determined that the Miner suffered from pneumoconiosis[1] and that he was totally disabled. The issue herein is whether his total disability was due to pneumoconiosis. Total disability due to pneumoconiosis requires that pneumoconiosis as defined in §718.201, be a substantially contributing cause of the miner's totally disabling respiratory or pulmonary impairment. Substantially contributing cause is defined as having a "material adverse effect on the miner's respiratory or pulmonary condition" or as "materially worsen[ing] a totally disabling respiratory or pulmonary impairment which is caused by a disease or exposure unrelated to coal mine employment." §718.204(c)(1)(i) and(ii). Absent a showing of cor pulmonale or that one of the presumptions of §718.305 are satisfied, it is not enough that a miner suffer from a disabling pulmonary or respiratory condition to establish that this condition was due to pneumoconiosis. See §718.204(c)(2). Total disability due to pneumoconiosis must be demonstrated by documented and reasoned medical reports. *Id.* Prior to the regulations being amended, the Seventh Circuit had held that pneumoconiosis must be a "*simple* contributing cause" of the miner's total disability (pneumoconiosis must be a necessary, but need not be a sufficient cause of miner's total disability). *Hawkins v. Director, OWCP*, 907 F.2d 697 (7th Cir. 1990).

Upon reviewing the medical opinion evidence of record, I find that it is sufficient to establish that the Miner's pneumoconiosis was in fact a contributing cause of his total disability. Significant to this decision, is the definition of pneumoconiosis.

Section 718.201 defines pneumoconiosis as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments arising out of coal mine employment. This

[1]In its brief, Employer contends that the issue of pneumoconiosis should be revisited. The undersigned declines to do so, however. For the reasons stated by Judge Mosser, and affirmed by the Board, the autopsy evidence supports a finding of pneumoconiosis.

definition includes both clinical or "medical" pneumoconiosis, and statutory or "legal" pneumoconiosis. Pursuant to 20 C.F.R. §718.201(a)(2), legal pneumoconiosis encompasses any chronic lung disease or impairment and its sequelae arising out of coal mine employment. A disease "arising out of coal mine employment" includes any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment. 20 C.F.R. §718.201(b). The regulations also acknowledge that pneumoconiosis is recognized as a latent and progressive disease which may first become detectable only after the cessation of coal mine dust exposure. 20 C.F.R. §718.201(c).

Dr. Gelhausen was a treating physician who also had the opportunity to review the autopsy evidence. In his opinion, the Miner had been totally disabled by pneumoconiosis during his lifetime, and the disease was also a factor in his death. Dr. Heidingsfelder, who performed the autopsy, found anthracosis to be present, further finding a severe chronic lung disease consistent with Black Lung. In 1988, Dr. Murthy was equivocal in his diagnosis of coal worker's pneumoconiosis, however, after having the opportunity to review the autopsy report, he opined that coal worker's pneumoconiosis was present and same was a contributing cause of death.

Dr. MacLennan, who treated the Miner shortly before his death, opined that he was disabled as a result of his coal worker's pneumoconiosis, and that it was a factor in the Miner's death. Dr. Long also found that the disease was a factor in the Miner's death. Finally, Dr. Cohen, who reviewed the medical evidence herein, determined that the Miner's pneumoconiosis was a contributing cause of his respiratory disability and death.

By contrast, several physicians who reviewed the medical evidence of record, found that pneumoconiosis was not a factor in the Miner's impairment or death. Thus, Dr. Crouch found idiopathic pulmonary fibrosis, specifically finding that pathologic changes of coal worker's pneumoconiosis were not present. Dr. Naeye also found that the characteristics of coal worker's pneumoconiosis were not present, specifically finding the disease to be absent. In his opinion, death was unrelated to occupational exposure to coal dust. In his deposition testimony, Dr. Naeye conceded that Dr. Heidingsfelder's assessment that anthracosis was present was accurate, but stated that it was not the same as a finding of coal worker's pneumoconiosis.

Dr. Selby, who had the opportunity to examine the Miner and review the autopsy findings, opined that the Miner did not suffer from coal worker's pneumoconiosis. In his opinion, the Miner's idiopathic pulmonary fibrosis was the cause of his death. Dr. Tuteur also found that coal worker's pneumoconiosis was not present. Dr. Wiot found the Miner to have been suffering from idiopathic pulmonary fibrosis.

With regard to those physicians who find that coal worker's pneumoconiosis was not present, despite the finding of anthracosis, I find that their opinions run contrary to the definition set forth under the Act and regulations. *See 20 C.F.R. 718.201; Peabody Coal Co. v. Director, OWCP*, 972 F.2d 178 (7th Cir. 1992); *Taylor v. Director, OWCP*, BRB No. 01-0837 (July 30, 2002 (unpublished). No only is anthracosis included in the definition of pneumoconiosis, but any pulmonary condition which is significantly related to, or aggravated by, coal mine dust exposure falls into that definition. For this reason, and given their failure to acknowledge that anthracosis is accepted for the purposes of the Act, as coal worker's pneumoconiosis, the opinions of Drs. Tuteur, Naeye and Crouch are not as persuasive as those of Drs. Heidingsfelder, Murthy, MacLennan, Long and Cohen.

Most persuasive to the undersigned is the opinion of Dr. Cohen, who reviewed extensive medical evidence, whose qualifications are excellent and whose reasoning as set forth above, is the most compelling. To claim that the Miner, who clearly had anthracosis, nearly forty years of coal mine employment, objective laboratory findings and findings upon physical examination which supported a finding of pneumoconiosis and total respiratory disability, was disabled as a result of a pulmonary condition whose etiology is unknown is just not persuasive.

As Dr. Cohen pointed out, "it makes little sense to attribute this patient's extensive pulmonary fibrosis to 'idiopathic' or unknown cause, when he has nearly 40 years exposure to a known cause of fibrosis, coal dust." The opinions of those physicians who conceded that the Miner did have anthracosis, but go on to find no coal worker's pneumoconiosis or disability due thereto, are outweighed for the reasons set forth above. Similarly, those physicians who find no evidence of any coal mine dust related disease or process to be present are not persuasive, given the autopsy report and indeed, the totality of the medical evidence. It should also be noted that several of the physicians who find the disease to be present and to have been disabling are board-certified, and were the Miner's treating physicians. *See* 20 C.F.R. Section 104(d). I find that their medical opinions are well-reasoned and well-supported by the objective laboratory data of record, as well as by the opinions of Drs. Heidingsfelder, Cohen, Long, and Combs.

Based upon the opinions of Drs. Cohen, Murthy, Gelhausen, MacLennan, and Heidingsfelder, I find that the Miner's pneumoconiosis was a contributing factor to his respiratory disability. It is within the province of the administrative law judge to weigh the medical opinion evidence, and he is "not bound to accept the opinion or theory of any medical expert." *Underwood v. Elkay Mining, Inc.*, 105 F.3d 946, 949 (4th Cir. 1997). The administrative law judge must examine the reasoning employed in a medical opinion in light of the objective material supporting that opinion, and also must take into account any contrary test results

or diagnoses. *See Director, OWCP v. Rowe*, 710 F.2d 251, 255 (6th Cir. 1983). In so doing, I find the reports of the aforementioned physicians to be worthy of greater weight, and find that they are sufficient to establish that the Miner's pneumoconiosis contributed to his disabling respiratory impairment. Thus, the Miner has not only established a material change in condition, but entitlement to benefits.[2] Next to be considered is the claim of his widow, the Claimant herein.

Death Due to Pneumoconiosis

The Claimant filed her survivor's claim on September 27, 1994. Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). As the existence of pneumoconiosis established in the miner's claim serves in Claimant's favor, the only issue left to be resolved with respect to her claim is whether or not the Miner's death was due to pneumoconiosis.

Section 718.205 provides that benefits are available to eligible survivors of a miner whose death was due to pneumoconiosis. An eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that the miner's death was due to pneumoconiosis;

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis.

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a substantially contributing cause of a miner's death if it hastens the miner's death. §718.205(c)(5). The circuit courts developed the "hastening death" standard, which requires establishment of a lesser causal nexus between pneumoconiosis and the miner's death. Thus, pursuant to *Peabody Coal Co. v. Director, OWCP*, *supra.*, pneumoconiosis is a substantially contributing cause of death if it hastens the miner's death in any way.

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[2]I do not find the medical opinion evidence submitted with the prior claim sufficient to outweigh the most recent evidence of record. While fully reviewed, it will not be detailed herein, but is incorporated by reference, as same was fully set forth by Judge Huddleston in his Decision and Order of October 2, 1985. (DX 51).

While the Seventh Circuit has held that it is inappropriate to give greater weight to the prosector merely because of his status as such, *Peabody Coal Co. v. McCandless*, 255 F.3d 465 (7th Cir. 2001), having taken this holding into account, I nonetheless credit the medical opinion of the prosector, Dr. Heidingsfelder, supported as it is by the opinions of Drs. Cohen, Long, Murthy and MacLennan, in determining that the Miner's pneumoconiosis did indeed, hasten his death. For the reasons set forth above, I find their opinions to be better-reasoned, more persuasive and worthy of greater weight. Accordingly, I find that death was hastened by pneumoconiosis pursuant to 20 C.F.R. Section 718.205(c)(5).

Entitlement

The Claimant has established that the Miner was entitled to benefits during his lifetime, augmented for one dependent. The Claimant has also established that she is entitled to survivor's benefits.

Date of Entitlement

Ordinarily, the date of commencement of benefits is determined by the date of onset of total disability. 20 C.F.R. 725.503; *Rochester & Pittsburgh Coal Co v. Krecota*, 868 F.2d 600 (3rd Cir. 1989). If medical evidence does not establish the date on which a claimant became totally disabled due to pneumoconiosis, then the claimant is entitled to benefits as of his filing date. This is the case unless there is medical evidence which, if credited indicates that the claimant was not totally disabled at some point subsequent to his filing date. *See Lykins v. Director, OWCP,* 12 BLR 1-181 (1989).

The Miner's claim was a duplicate claim wherein a "material change in condition" was established. Once a material change in condition is demonstrated, the subsequent claim is to be considered a new and viable claim. Therefore, the filing date of the subsequent claim determines which substantive regulations apply as well as the earliest date from which benefits may be awarded if the miner is found to be entitled. *Spese v. Peabody Coal Co.*, 11 BLR 1-174, 1-176 (198)*, dismissed with prejudice,* Case No. 88-3309 (7th Cir. Feb. 12, 1989)*(unpub.). See also Peabody Coal Co. v. Spese,* 117 F.3d 1001 (7th Cir. 1997)*(en banc)* (the earliest date of onset in a multiple claim under Section 725.309 is the date on which that claim is filed; the claim does not merge with earlier claims filed by the miner).

The record in this case does not contain medical evidence establishing exactly when the Miner became totally disabled. Therefore, payment of benefits should begin as of May 1, 1988, the first day of the month in which the Miner filed his duplicate claim.

Attorney's Fees

No award of attorney's fees for services to the Claimant is made herein as no application has been received. Thirty days is hereby allowed to the Claimant's counsel for the submission of such an application and his attention is directed to Sections 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. Parties have ten days following receipt of any such application within which to file objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

It is hereby ORDERED that the Employer, Old Ben Coal Company shall:

1. Pay to the Estate of Hugh Hyslop all benefits to which the Miner was entitled commencing as of May 1, 1988 until the month prior to his death on September 3, 1994;

2. Pay the Claimant, Bernice Hyslop, all benefits to which she is entitled under the Act commencing as of the month in which the Miner died; and

3. Pay the Claimant's attorney fees and expenses to be established in a supplemental decision and order.

CLEMENT J. KICHUK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.